ENVIRON

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Ms. Laura Siclari
Hoagland, Longo, Moran, Dunst, & Doukas, LLP
40 Paterson Street, PO Box 480
New Brunswick, NJ 08903

Subject: **Jack Nacht vs. Mannington Mills, Inc.**
Index no.: 114274-06

Dear Ms. Siclari:

Thank you for referring the above matter to me for epidemiological assessment. It is my understanding that the Plaintiff claims that Mr. Nacht's pleural mesothelioma resulted from exposure to asbestos from the handling of flooring products allegedly obtained from Mannington Mills, Inc.

Following is my expert report in this matter, detailing my credentials, the methods used and materials relied upon, a critical review and synthesis of the relevant epidemiological literature, application of the scientific assessment to the facts apparent in this case, as well as my scientific opinions.

INTRODUCTION

Qualifications

I am by training and experience an epidemiologist. I was trained at the Master's level at the School of Public Health, University of Massachusetts and at the doctoral level at the School of Public Health, University of North Carolina. For ten years I served first as Assistant Professor and then as Associate Professor of Epidemiology in the Department of Biostatistics and Epidemiology of the School of Public Health and Health Sciences, University of Massachusetts. In 1991, I founded Applied Epidemiology, Inc., Amherst, Massachusetts, which in November 2003 merged with ENVIRON International Corporation, where I am a Principal, and serve as Director of Epidemiology. I have special interest and experience in matters pertaining to workplace exposures to various materials and chemicals including asbestos.

I have extensive experience in, for example, designing, conducting and publishing primary epidemiological research; critical review and synthesis of published

epidemiological literature; the graduate training of epidemiologists, including classroom teaching, advising and chairing of Master's and Doctoral Committees; and serving in advisory, review and editorial capacities at the local, national and international level. I serve as Adjunct Associate Professor in the Department of Epidemiology, University of North Carolina at Chapel Hill, and Adjunct Associate Professor at Georgetown University, where I have team-taught on several occasions "Epidemiological Applications to Population Health" in the School of Nursing and Health Sciences. I am also an Adjunct Associate Professor and member of the Dean's Advisory Board of the School of Public Health and Health Sciences, University of Massachusetts, where I teach on occasion. I am a Fellow of the American College of Epidemiology, and serve as Vice Chair of the College's Finance Committee. I also serve on the editorial board or as a reviewer of several scientific journals.

I have taught methods for critical review and synthesis of epidemiological studies as part of the core curriculum for Masters and Doctoral candidates in Public Health, and have used these methods to evaluate associations between various exposures and health outcomes. I have applied the same critical review approach to my evaluation of the epidemiological literature on occupational asbestos exposure and risk of mesothelioma, which is summarized below.

A copy of my current Curriculum Vitae which provides additional details as well as a list of my publications is attached.

Overview of approach

I have evaluated the peer-reviewed, published epidemiological literature on the relationship between exposure to various types of asbestos and the risk of mesothelioma, including the consideration of its very long latency (time between exposure and disease occurrence), to determine whether exposure to flooring materials alleged to have been obtained from Mannington Mills was likely to have caused Mr. Nacht's pleural mesothelioma. I have employed standard and widely accepted methods for critically and comprehensively reviewing and synthesizing the published, peer-reviewed epidemiological literature, and formulated my scientific opinions and conclusions based on this analysis. In addition to the relevant peer-reviewed, published epidemiological literature, I draw upon my education, training and professional experience, as well as on my analysis of materials provided to me by counsel for Defense, including but not limited to the complaint, interrogatory responses and deposition transcripts, to formulate my professional opinions and the conclusions offered. A bibliography of all materials relied upon is included below. I expect to review and comment upon additional scientific publications, documents, testimony, expert reports, exhibits and discovery related to the topics of this report as they become available.

METHODS

The epidemiological approach

Epidemiology is the field of public health that includes the study of incidence, prevalence, and distribution of disease in human populations, and factors that may be related to disease occurrence. It is a science that employs standard methods to identify and interpret statistical correlations, called "associations," between disease occurrence and other factors. Epidemiological research results are central to the determination of the role of specific risk factors in the general causation of disease in humans, and are broadly relied upon by epidemiologists and other professions as a basis for decision-making, including development of policy and judgments regarding specific causation. The validity of the epidemiological evidence and the validity of its interpretation determine the reasonableness of causal judgments that rely upon such evidence.

Epidemiological research addresses whether a disease is associated with specific exposures in groups of people or populations. Exposures measured on an individual basis provide the strongest evidence of an association, if it exists, between the exposure of interest and disease. Analytic techniques also can control for, or eliminate possible effects of other exposures that may be related to the exposure of interest, or the disease under investigation.

Epidemiologists are generally concerned with the impact that bias, due to systematic error, might have on study results. Systematic error in the methodology or due to missing or inaccurate information can render results invalid or even misleading, possibly related to how the groups being compared have been defined. Factors determining the quality of epidemiological studies include the ability to avoid biases such as selective participation of certain subsets of individuals (selection bias), systematic errors in responses or measured data (information or misclassification bias), and identification of other strong risk factors for the same outcome that are correlated with the factor of interest (confounding bias). Though the potential for bias exists in all studies, some study approaches and research settings are more prone to bias than others. The degree to which these challenges are addressed and overcome in an epidemiological study partly determines the strength and validity of the study results.

Interpretation of the epidemiological literature also considers the role of chance in the results. Statisticians and epidemiologists evaluate the probability that an observed result is due to chance by applying tests of statistical significance. If the results are not statistically significant, chance cannot reasonably be ruled out as an explanation for the reported association (i.e., accepting a 5% error rate).

Dose-response assessment, or the evaluation of the relationship between estimated or actual dose of exposure and the disease risk, is a key tool of epidemiology. Ideally, the dose estimate is derived based on a reasonable period of disease latency. Latency is usually described as the time elapsed between the first known exposure to the agent of interest (often indicated by date first employed in a particular job where exposure is likely) and the diagnosis of the disease of interest. This is also known as maximum latency, and is influenced by the ability to validly identify first exposure as well as the

ability to detect the disease soon after it occurs. For many cancers that produce solid tumors, the latency is usually 20 or more years, and can exceed 50 years (as with mesotheliomas). If exposure is assessed without regard to disease latency, some or all of the exposure evaluation may be irrelevant to the occurrence of the disease.

Assessments of dose-response and latency are frequently used to inform causal judgment and policy formulation. The greatest impediment to such analyses is that many epidemiological studies fail to, or cannot, accurately characterize first possible exposure and the specific level or concentration of exposure for each individual in the study: in fact, most studies that consider exposure rely upon surrogate measures such as employment history.

In addition to assessing the methodological quality of individual studies, weight of evidence syntheses consider the overall breadth and quality of the literature available. Just as individual studies might be subject to systematic biases, a body of literature might be biased because of its focus on a chosen or preferred research hypothesis or study approach, or as a result of selection for publication. Such "publication bias" occurs when authors preferentially submit, and journals preferentially accept, studies demonstrating positive findings, even if such studies may be positive due to methodological weakness (e.g., small study size) or errors (e.g., exposure misclassification). Null study findings, even if based on well-designed and conducted studies, are considered "less interesting" and are less likely to be published (Hennekens and Buring 1987). The overall bias will be exaggerated if null findings contradict positive findings and/or if a weak study supports (or replicates) previously "accepted" associations.

Use of epidemiology for judgments of disease causation

Where a balanced and complete literature is available, it is possible to characterize that literature as reasonably establishing an association that is or is not probably causal. If the weight of evidence favors a judgment of general causation, i.e., that a risk factor under certain conditions is capable of causing the disease in adequately exposed persons, epidemiological research can further help determine whether specific exposure attributes and other risk factors are more or less likely to contribute to risk among exposed persons. These risk factors may include but are not limited to the following:

- Dose or exposure concentration
- Type or form of exposure (chemical composition or physical structure)
- Timing of exposure (year first exposed, duration of exposure, etc.)
- Host susceptibility (genetic polymorphisms)
- Host attributes (age, sex, ethnicity, etc.)
- Co-exposures (viruses, smoking, etc.)
- Co-morbidity

Epidemiology generally cannot directly ascertain specific causation, i.e., whether a specific exposure played any role in the disease of specific individuals. Some cases of disease are idiopathic, and arise regardless of an individual's exposure history. However, understanding at a group level those factors that are associated with increased risk of disease may improve our ability to identify high or higher risk groups so that reductions

in exposure can be targeted and implemented to prevent future disease. Based on these methods, we may also improve our ability to determine which subgroups are at increased risk due to their exposures, and by extension (and for non-scientific purposes such as decision-making and litigation) determine that the relationships observed at the group level may reasonably be applied to individuals who are in the study group or an adequately similar population. The determination of causation, however, remains a judgment, and cannot be proven. Although direct experimental evidence may offer stronger support for causal inference, experiments on health effects of toxic materials on humans are unethical. Toxicological or other laboratory data collected from animal models are useful for understanding the mechanisms of effects and for developing hypotheses regarding human health effects, but are not directly applicable to humans. For these reasons, well-designed epidemiological studies have been identified as the preferred scientific support for regulatory agencies such as the Environmental Protection Agency (EPA) and the Occupational Safety and Health Administration (OSHA), which determines policies regarding possible adverse effects of exposures. For the same reasons, the determination that an association is causal in humans is most reasonably based on the availability of and proper evaluation of good epidemiological data.

Epidemiological review of asbestos and malignant mesothelioma

For this report, PubMed was used to identify key research reports and reviews published in the peer-reviewed medical/health literature that are relevant to the topics of this case. Provided by the US National Library of Medicine, PubMed is a powerful standard research tool available free over the internet, which searches the medical literature published since about 1966. The keywords "asbestos," "crocidolite," "amosite," "amphibole," "chrysotile," "pleural," "mesothelioma," "occupation," "dust," "floor installation," "floor removal," "floor mechanic," "floor tile," "floor covering," "vinyl asbestos tile," and "exposure assessment" were entered in various forms and combinations to identify a universe of potentially relevant articles available in the English language. Abstracts of the articles identified were screened for relevance, and the full article for those meeting at least minimal criteria (epidemiological study, standard methods utilized, study population asbestos-exposed, analyses conducted by occupational risk factors such as asbestos exposure level, duration of exposure, type of industry, type of asbestos present, or other risk factors that help explain the risk of mesothelioma) were more thoroughly evaluated. Occupational studies of the primary and secondary asbestos industries were included and are summarized below, as these studies examine populations of workers with documented and often heavy asbestos exposures and provide a considerable basis for understanding and evaluating potential risks among groups with lower exposures, including presumed and background level exposures.

EPIDEMIOLOGY OF ASBESTOS AND MALIGNANT MESOTHELIOMAS

Overview

Malignant mesotheliomas are cancers that arise most often in the pleura, the thin layer of tissue that surrounds the lungs and lines the chest cavity, or the peritoneum, the thin layer of tissue that surrounds the abdominal cavity and lines the abdominal organs (Blot and

Fraumeni 1996). The risk of all types of mesothelioma combined is about 11 per million population per year in the US, with about 3,000 new cases diagnosed each year (Antman, Hassan et al. 2005). The incidence of cases not thought to be due to occupational exposure has been estimated at 1-2 cases per million person years (Bertazzi 2005).

Tumor registry data show an increase in the number of all types of malignant mesotheliomas, combined, beginning in the 1950s (Blot and Fraumeni 1996). Incidence increased sharply beginning in the 1970s and continued to rise through the mid-1990s (Price and Ware 2004; Bertazzi 2005). The increase has occurred almost exclusively among white men, as the rate for women in the U.S. has been stable since the 1970s at about 5 per million per year. Rates for non-white men have also remained relatively stable over time (Bertazzi 2005).

Risk factors for mesotheliomas include exposure to amphibole asbestos, erionite (a fibrous mineral from the zeolite group) and thorium dioxide (Thorotrast) – a radioactive contrast medium used in X-ray diagnostics until the 1950s in the U.S. "Amphibole" refers to a group of five of the six fibrous minerals that are considered to be asbestos: crocidolite, amosite, anthophyllite, actinolite and tremolite. Amphiboles are characterized by thin, rod-like fibers. In contrast, the most widely used form of asbestos, chrysotile, is a serpentine mineral, with curly and pliable fibers. Simian Virus 40 (SV40) cells have been discovered in mesothelioma tumors, suggesting the virus might play some role in the development of mesothelioma (Carbone, Rizzo et al. 2000; Vilchez, Kozinetz et al. 2003). Neither smoking nor ionizing radiation is considered a risk factor for mesothelioma (Antman, Hassan et al. 2005).

Some mesotheliomas are idiopathic, meaning they are not due to exposure to any of the recognized risk factors for the disease. Estimates of the rate of idiopathic mesothelioma may be derived from population-based cohort studies, where between 10% and 50% of mesotheliomas detected occurred in the absence of identifiable asbestos exposure (Boffetta 1998; Antman, Hassan et al. 2005; Bertazzi 2005). A proportion of all occupationally asbestos-exposed individuals with mesothelioma are also believed to develop mesothelioma due to causes other than their occupational asbestos exposure. Some number of idiopathic cases is expected to occur even among individuals with a history of substantial amphibole asbestos exposure.

Asbestos exposure and risk of pleural mesothelioma

Among asbestos-exposed persons, the chief factors that influence the risk of pleural mesothelioma are asbestos fiber type, the time since first exposure to asbestos (latency), and exposure (dose).

Exposure to amosite, crocidolite or other types of amphibole asbestos is the clearest risk factor for pleural mesothelioma. In contrast, epidemiological evidence suggests the association between chrysotile asbestos exposure and pleural mesothelioma risk, if any, is weak.

The epidemiological literature indicates that average latency – i.e., the time between first adequately high exposure to amphibole asbestos and occurrence of mesotheliomas – is

very long, and may be as much as 60 years. The duration of latency may depend on both dose and fiber type. Low exposures are generally associated with longer latencies. Occupational studies of amphibole exposure and mesotheliomas typically show a dose-response relationship, with low or no risks observed among those with the lowest exposures.

Low-level asbestos exposures are also ubiquitous in the general population (i.e., not occupationally exposed persons), and can arise from regional geological features and from fibers released into the environment from consumer and building products. Use of asbestos (predominantly chrysotile) -containing consumer and building products increased after World War II through the 1970s, at which time a ban went into effect on asbestos in some consumer products, and asbestos was voluntarily withdrawn from other products. Ambient asbestos exposures also are thought to have increased and then declined over this interval, parallel to the increase and decrease in use of asbestos-containing consumer goods, though ambient environmental levels remained far below those measured in some work places. The lack of an increase over time in mesothelioma risk among women and non-white men has been taken as evidence for a necessary threshold, greater than ambient environmental levels, for amphibole asbestos exposure to lead to increased risk of mesothelioma (Bertazzi 2005).

Occupational studies of primary asbestos exposure and mesothelioma

All primary asbestos industries have reported increased mortality from pleural mesothelioma or all types of mesothelioma, combined. The primary asbestos industries include **mining and milling** (de Klerk, Armstrong et al. 1989; Piolatto, Negri et al. 1990; Sluis-Cremer, Liddell et al. 1992; Liddell, McDonald et al. 1997), **asbestos cement manufacturing** (Thomas, Benjamin et al. 1982; Finkelstein 1984; Alies-Patin and Valleron 1985; Ohlson and Hogstedt 1985; Gardner, Winter et al. 1986; Hughes, Weill et al. 1987; Raffn, Lynge et al. 1989; Albin, Jakobsson et al. 1990; Neuberger and Kundi 1990), **textile manufacturing** (McDonald, Fry et al. 1983a; McDonald, Fry et al. 1983b; Peto, Doll et al. 1985; Dement, Brown et al. 1994), **insulating** (Seidman, Selikoff et al. 1986; Seidman and Selikoff 1990), **friction and insulation materials manufacturing** (McDonald, Fry et al. 1984; Enterline, Hartley et al. 1987; Newhouse and Sullivan 1989); and **filter assembly and manufacturing** (McDonald, Gibbs et al. 1978; Jones, Smith et al. 1980; Talcott, Thurber et al. 1989) . Studies conducted in the primary asbestos industries, where the broadest range of exposures can be found, offer the best opportunity to quantify mesothelioma risks.

Mesothelioma risk depends on asbestos fiber type

Chrysotile accounts for 95% of asbestos produced in the world (Harington 1991; Terracini 2006) and it is the most commonly used asbestos fiber in the US. As noted above, however, risk of mesothelioma is much more strongly associated with amphibole asbestos exposure. Among the amphiboles, crocidolite is most strongly associated with mesothelioma risk. Intermediate risks are seen among workers exposed to amosite. Increased risk of mesothelioma from occupational exposure to chrysotile alone is less apparent and not seen in many studies. The risk differential for mesothelioma at similar exposure levels of chrysotile: amosite: crocidolite has been estimated to be 1:100:500

(McDonald and McDonald 1996; Hodgson and Darnton 2000). In studies where mesothelioma risk appears to be increased among workers with high-level chrysotile exposures (e.g., asbestos miners), there is reasonable evidence that the risk derives from contamination by amphibole fibers, as comparable groups heavily exposed to pure chrysotile appear not to be at increased risk of mesothelioma (McDonald and McDonald, 1996).

Crocidolite asbestos

In employees with predominantly or heavy crocidolite exposure, the relative risk of pleural mesothelioma compared to unexposed or less exposed employees is extremely high, even while the absolute risk remains low. For example, proportional mortality from mesothelioma was examined among 33 men employed during 1953 in a Massachusetts factory that manufactured cigarette filters containing crocidolite. Through 1988, a total of 28 deaths had occurred. Five were from pleural mesothelioma, with only 0.01 expected (RR=460; 95% CI 150, 1080) (Talcott, Thurber et al. 1989). Jones et al. investigated causes of death among 951 women who had used crocidolite to manufacture military and commercial grade gas mask filters during the 1940s. The authors reported 29/166 deaths were due to mesothelioma, with risk positively associated with duration of exposure. The most deaths in this group occurred during the middle 1970s, approximately 30 years after first exposure to crocidolite (Jones, Smith et al. 1980).

Initial reports for a cohort of 13,450 friction product workers who primarily manufactured brakes and brake material between 1941 and 1979 identified 10 deaths from mesothelioma, 9 among those with known crocidolite exposure. There was no excess among those exposed to chrysotile, the predominant fiber type used at the factory (Newhouse, Berry et al. 1982; Berry and Newhouse 1983). An updated mortality study of the cohort reported a total of 13 mesothelioma deaths, 11 among those known to be exposed to crocidolite (Newhouse and Sullivan 1989).

Chrysotile asbestos

Studies of workers predominantly exposed to chrysotile fibers inconsistently demonstrated slightly elevated risks of mesothelioma mortality compared to non-exposed populations. McDonald et al. identified 4,137 male textile workers who were mainly exposed to chrysotile who were employed for more than one month between 1938 and 1958 at a plant in Pennsylvania. Fourteen out of 1,392 death certificates mentioned mesothelioma (McDonald, Fry et al. 1983b). Dement et al. (1994) reported mortality for a similar South Carolina textile manufacturing plant cohort, including those employed for at least one month between 1940 and 1965, with vital status ascertained through 1990 (Dement, Brown et al. 1994). Two pleural mesothelioma deaths were reported among workers primarily employed in the spinning department, with latency of 37 and 34 years, and 25 and 32 years employment, respectively. A third pleural mesothelioma death was identified when mortality follow-up was extended through 2001. The most recent decedent also had been employed in the spinning department, with a latency interval of nearly 50 years (Hein, Stayner et al. 2007).

Among 1,058 Italian chrysotile asbestos miners employed since 1946, two deaths due to pleural mesothelioma were identified among 427 total deaths – one in a worker with 20 to 30 years since first exposure and the other in a worker with more than 30 years since

first exposure (Piolatto, Negri et al. 1990). One pleural mesothelioma was reported in a study of 2,167 chrysotile asbestos cement workers (Gardner, Winter et al. 1986) and no mesothelioma deaths were reported by McDonald et al. (1984) among 3,641 US friction product workers where chrysotile was the predominant fiber used (McDonald, Fry et al. 1984).

It remains unclear whether the occasional mesothelioma cases seen among heavily chrysotile-exposed workers are due to high-intensity chrysotile fiber exposure, amphibole fiber contaminants, unrecognized amphibole exposure from other sources, or are of idiopathic origin.

Mixed fiber types

Workers handling primarily one type of asbestos may unwittingly have exposure to other types, or may come to a specific workplace with prior exposure to amphibole asbestos. Also, as mentioned above, chrysotile may be contaminated with amphibole fibers. When mesothelioma risk among primarily chrysotile-exposed occupational cohorts has been identified, it has generally not been possible to determine if the risk was due to chrysotile, contamination with amphiboles, other sources of amphibole exposure, or from some combination of these (Elmes 1994; Britton 2002). For example, the Pennsylvania textile workers described above were mainly exposed to chrysotile, but crocidolite and amosite were also present in the plant. Although information about exposure to specific fiber types was not provided in the report, the authors attributed the mesothelioma cases to amphibole exposure (McDonald et al., 1983b).

Prior to World War II, insulation workers in New York and New Jersey were primarily exposed to chrysotile fibers. Subsequently, amosite was added to some of the insulation. In a cohort study of New York and New Jersey insulators, those working for long periods after the war were most likely exposed to amosite, an amphibole, and were at substantially increased risk of mesothelioma. No increase in risk was observed among those only exposed to chrysotile prior to the war, or those exposed to both chrysotile and amosite after the war but for less than 20 years (Selikoff, Hammond et al. 1979). This latter observation suggests that some exposure threshold (concentration and duration) may be required before risk is meaningfully elevated; however, evidence of such a threshold has not been consistently observed.

Yarborough also reviewed studies of occupational cohorts with exposures to mixtures of fiber types (Yarborough 2006). The cohorts, including some from the studies cited above, had a total of 32,000 employees with exposures thought to be relatively pure chrysotile. Among these, only seven mesothelioma cases were identified, and Yarborough found reason to question the accuracy and adequacy of the exposure evaluation and/or diagnosis for each of them.

Lung fiber burden analyses generally indicate exposures to mixtures of fiber types, even if work histories are unable to document mixed exposures (Yarborough 2006). For example, McDonald et al., (1997) examined dried lung specimens from chrysotile miners and millers in Quebec who died of mesothelioma. Of the 27 mesothelioma cases, 21 dried lung specimens were available. The investigators found tremolite and chrysotile fibers in 14 specimens from cases who worked in the Thetford mines, and chrysotile,

tremolite, crocidolite and amosite in specimens obtained from the remaining 7 cases who worked in the mines in Asbestos (McDonald, Case et al. 1997). This investigation indicates that mesotheliomas that were attributed to chrysotile exposure may be due to the amphiboles contaminating the chrysotile at specific mining sites.

Although they offer suggestive evidence, lung fiber burden studies can be criticized on the grounds that fibers found in the lung at autopsy have an unknown relationship to fiber exposures that actually led to mesothelioma induction. Epidemiological evidence suggests that at least 30 years, and perhaps as much as 50-60 years, must elapse between initial exposure and the onset of mesothelioma (see discussion of latency, below). Furthermore, amphiboles are expected to be found many years after exposure has ceased due to their persistence in lung tissue, whereas chrysotile fibers are relatively rapidly broken down and eliminated (Churg and DePaoli 1988; Britton 2002; Bernstein and Hoskins 2006). It is the relative durability and persistence of the amphiboles that is believed to contribute to their carcinogenic potency.

Exposure (Dose)

The relationship between mesothelioma and either intensity or duration of exposure, or a cumulative (intensity and duration combined) estimate, is complex. Because exposure measurements are frequently unavailable in epidemiological studies, duration of employment or other surrogates are often used as indicators of "dose." Occupational studies that adequately account for disease latency and fiber type generally report a positive association between mesothelioma risk and either duration of employment or quantitative dose estimates. For example, of 8,009 deaths investigated among workers in the asbestos mines in Quebec, 22 of 25 mesothelioma cases were among men employed 20 or more years in the Thetford mines, and 5 additional cases were among men employed for at least 30 years in the Asbestos mines (McDonald et al., 1997). In a later publication, it was reported that the rate of mesothelioma increased with increasing fiber-years of exposure (Liddell, McDonald et al. 1997). Among 6,506 crocidolite miners/millers in Western Australia, the relative risk was 10.5 (95% CI 3.12-35.1) for pleural mesothelioma among those with more than 6 months of exposure compared to those with shorter duration of exposure (de Klerk, Armstrong et al. 1989).

Among asbestos cement workers, Albin et al (1990) reported an increasing risk of pleural mesothelioma with increasing cumulative exposure compared to unexposed workers: RR=1.9 for workers with an average dose of 3.1 f/ml-years; RR=21.2 for an average dose of 25.6 f/ml-years; and RR=22.8 for average dose of 88.2 f/ml-years (Albin, Jakobsson et al. 1990). Similarly, Finkelstein (1984) reported an increasing trend in mesothelioma (all types) mortality with increasing dose among asbestos cement workers. The mean cumulative exposure for eleven pleural mesothelioma cases was 42 f/ml-years, and 161 f/ml-years for eight peritoneal mesothelioma cases (Finkelstein 1984). Among amosite-exposed factory workers, the lower the dose (time worked), the longer the time required for development of disease (Seidman, Selikoff et al. 1986).

Fiber type remains an important consideration when assessing the risk of mesothelioma associated with exposure concentration or duration. Among a cohort of South African miners, workers were exposed to crocidolite, amosite, or mixtures of both fibers (Sluis-

Cremer, Liddell et al. 1992). Of the 30 cases of mesothelioma identified, 20 occurred among those with crocidolite exposure (none with less than 12 months exposure), 4 among those exposed to amosite for more than 3 months, and 6 among those with mixed exposures for more than 3 months. Cumulative exposure averaged 15.2 fibers/ml/year for the amosite group compared to 9.6 fibers/ml/year for the crocidolite group.

Time since initial exposure (Latency)

Time since initial exposure to amphibole asbestos is a strong predictor of incidence of mesothelioma. In the PMR study of cigarette filter manufacturers described above, the median interval from first exposure to crocidolite asbestos until mesothelioma death was 34 years (range, 26 to 37) (Talcott, Thurber et al. 1989). Any mesothelioma deaths that occurred after 1988 would only increase the average estimated latency, as cases with longer latency would not yet have been detected as of 1988, when the study was completed. In general, when mortality is used instead of disease incidence or diagnosis, the overall duration of latency will be somewhat exaggerated. Because the average survival time after mesothelioma diagnosis is short (about 1 year) (Stewart, Edwards et al. 2004; Antman, Hassan et al. 2005), the effect of using death rather than incidence or diagnosis to calculate estimated latency for mesothelioma will be limited.

In Australia, the incidence of malignant mesothelioma lagged 20 to 30 years behind trends of amphibole (primarily crocidolite) exposure. Incidence of mesothelioma for 1964 to 1968 among those 35 years or older at diagnosis was less than 1.0 case per million person-years, increasing to 15.5 cases per million person-years in 1979-1980. Among men aged 65 to 74 in 1979-1980, mesothelioma occurred in 69.7 cases per million person-years (Musk, Dolin et al. 1989).

Among 10,918 chrysotile miners and millers in Quebec first employed in 1904 and followed until 1992, a total of 38 mesotheliomas were reported – 33 in miners and millers and 5 in factory workers. Of these, 21 occurred in workers from the Thetford mines and in Asbestos mines and mills – both sites among the earliest operations in Quebec (Liddell 1997). Average latency was 47 years, with a range of 21 to 60 years. PMRs increased with year of death: no mesothelioma deaths occurred before 1950, but subsequent mesothelioma death rates were 0.18% (1950-1974), 0.68% (1975-1984) and 1.10% (1985-1992). Rates of mesothelioma were significantly greater at the oldest Thetford mines (35.3/100,000 subject years) than at the Asbestos mine and mills (13.2/100,000) (McDonald, Case et al. 1997), possibly because of the longer time follow-up time.

A mortality study of 4,137 textile manufacturing workers employed more than one month between 1938 and 1974 and exposed to chrysotile, amosite and crocidolite identified 14 mesothelioma deaths between 1960 and 1975. With the exception of one subject, deaths occurred 25 to 53 years from first employment (McDonald, Fry et al. 1983a).

Initial studies of the insulation workers in New York and New Jersey followed through 1976 reported no mesotheliomas among those with less than 20 years exposure; 7 mesotheliomas occurred among those with 20 to 34 years, and 31 mesothelioma cases in New York/New Jersey insulators occurred after 35 years of exposure. Among a larger cohort of US and Canadian insulation workers followed through the same period, only 5

of 224 mesotheliomas occurred between 5 and 19 years after onset of exposure; rates of pleural mesothelioma were 2.78 per 1,000 person-years for 35 to 39 years after onset and 5.47 per 1,000 person-years for peritoneal mesothelioma 45 or more years after exposure onset. Additional follow-up of the 17,800 asbestos insulation worker cohort found peak mortality from mesothelioma (5.1 per 1,000) occurred 45 years after first employment. Although excess deaths from all causes combined decreased over time, there was no apparent decrease in deaths due to pleural mesothelioma or for those with more than 40 years since onset of first exposure (Seidman and Selikoff 1990).

Based on these studies, it is apparent that the greatest risk of mesothelioma occurs on average 40-50 years after first substantial exposure to amphibole asbestos occurs. Lower exposure concentrations would be expected to require even longer latency.

Studies of secondary asbestos exposure and mesothelioma

Construction trades

Mesothelioma risk has been described among employees in various construction trades due to their use of asbestos-containing insulation and building materials. Historically, exposure to asbestos in the building / construction industry occurred during installation and removal of insulation, installation of duct work for ventilation, among plumbers and electricians cutting into or disrupting installed asbestos insulation, and during plaster preparation/mixing and dry sanding. More recently, chrysotile asbestos exposures in construction may occur – but at much lower levels and almost entirely limited to chrysotile fibers – in the process of handling and installing drywall joint compound, roofing and siding materials, as well as some flooring materials. Higher exposures, and therefore greater mesothelioma risks, are likely to result from the removal, demolition or rehabilitation of older construction containing asbestos insulation or other friable amphibole-containing materials than in the installation of new, non-friable chrysotile-containing materials such as roofing and flooring materials, etc. Many building/construction products contained small amounts of asbestos until the late 1970's and early 1980's, when manufacturers began reducing and eventually eliminating asbestos in their products. Subsequently, the likelihood of asbestos exposure due to the use and handling of new building materials has been greatly reduced. The specific asbestos fiber type that may have been used in a particular product is often unknown (Huncharek 1992).

Several studies have reported risk of mesothelioma among construction workers (Robinson 1995; Wang, Dement et al. 1999; Stern, Lehman et al. 2001; Koskinen, Pukkala et al. 2002; Engholm and Englund 2005). For example, Robinson and colleagues (1995) studied the mortality experience of 61,682 men who died between 1984 and 1986 in 19 states who had been employed in various occupations in the construction industry, using usual occupation as reported on the death certificate. Cancers of the pleura and peritoneum were observed for carpenters (PMR=163; 95% CI 89, 274, based on 14 deaths), plumbers (PMR=327; 95% CI 106, 763, based on 5 deaths), insulation workers (PMR=2467 based on 2 deaths), and electricians (PMR=331; 95% CI 122, 719 based on 6 deaths). The largest subgroup of the cohort was construction laborers, with nearly 10,000 men included; although other causes of death were elevated in this subgroup, there were no mesothelioma deaths noted (Robinson 1995).

Koskinen et al. (2002) followed 16,696 Finnish construction workers participating in cancer screening campaign from 1990 through 2000. Of 1,320 incident cancers, 13 mesotheliomas generated roughly a doubling of risk (SIR=1.96; 95% CI 1.04, 3.35). The only occupational sub-groups with significantly elevated risks included insulators and electricians, with 3 and 4 mesotheliomas in each group, respectively (Koskinen, Pukkala et al. 2002).

Flooring mechanics

Epidemiological studies of construction industry employees include floor installers and removers. When construction occupations have been examined separately by trade, floor installers and removers have not been identified among those at increased risk of mesothelioma (Robinson 1995; Wang, Dement et al. 1999; Koskinen, Pukkala et al. 2002; Engholm and Englund 2005). Specific studies of mesothelioma or other asbestos-related risks focusing exclusively on floor installers and removers have not been identified.

Some estimates of exposure during standard floor removal procedures are available, and indicate very low fiber concentrations. Lange et al (1996) reported that exposures due to floor tile removal resulted in arithmetic and geometric mean airborne fiber concentrations 0.005 f/cm^3 (range: 0.005 to 0.01 f/cm^3) (Lange, Lange et al. 1996). Similarly, Williams and Crossman reported fiber concentrations resulting from breaking vinyl asbestos floor tiles with a hammer were at or below detection limits for OSHA-specified monitoring methods (i.e., exposures were <0.0058 str/ cm^3) (Williams and Crossman 2003).

Summary of occupational studies of secondary asbestos exposure in construction trades

Construction workers may be exposed to low levels of asbestos intermittently over a long period of time (the duration of their employment), and have been observed to be at low risk of mesothelioma. The specific exposure levels depend on trade, and occupations within the construction industry can be roughly ranked according to potential exposure. Plumbers and electricians tend to be most highly exposed; those involved with installation and removal of asbestos containing roofing and siding products are generally at lower risk, followed by wallboard installers and plasterers. Flooring mechanics typically do not demonstrate high levels of asbestos exposure and have not been reported in the occupational health literature to have elevated mesothelioma risk. The vast majority of asbestos used in construction materials is chrysotile, which has not been convincingly associated with risk for mesothelioma. The duration of the latency interval appears to be proportional to the intensity of amphibole exposure, with lower-intensity exposures requiring latency intervals of as much as 50 years or longer.

Although amphibole asbestos exposure is the most likely risk factor for mesothelioma, information on prior asbestos exposures of any kind may be sought specifically when mesothelioma is diagnosed. Information on specific occupational and other sources of asbestos exposure may be less actively sought for diseases or causes of death that have not been strongly associated with a specific exposure. Actively searching for any source of asbestos exposure when a diagnosis of mesothelioma is generated, or differentially seeking such information from cases and controls, would erroneously create or exaggerate observed associations between mesothelioma and asbestos exposure. These

forms of bias may most impact studies of occupational groups less clearly or consistently exposed to amphibole asbestos.

SUMMARY OF RELEVANT FACTORS IN THIS CASE

Mr. Nacht was diagnosed with pleural mesothelioma in August 2006. Based on an extensive and critical evaluation of the epidemiological literature, the main determinants of risk of pleural mesothelioma include exposure to amphibole asbestos fibers, with risk dependent both on exposure intensity and latency, with "latency" defined as the time between first exposure to substantial amphibole asbestos and the diagnosis of disease. Intermediate risks are noted among those exposed to mixed fiber, presumably in proportion to the concentration of amphibole asbestos fiber present. However, the literature also indicates that mesotheliomas occur in substantial proportion among persons with no significant exposure to amphibole asbestos fibers. These cases may be idiopathic or the result of unrecognized exposures that might have occurred 40-60 years prior to diagnosis. Understanding these factors – the intensity, duration and timing of exposure to specific asbestos fiber types – as they pertain to Mr. Nacht's exposure history and disease occurrence is important in evaluating whether specific potential sources can reasonably be considered causes of his mesothelioma.

According to his deposition testimony, Mr. Nacht was exposed to asbestos in the course of operating his retail carpet and floor tile business from 1946 until his retirement in 1997. The exposures he alleged were due to the asbestos content of certain of the floor coverings that he sold. Mr. Nacht testified in deposition that he was exposed to dust from floor tiles while he unpacked them and when he broke tiles to demonstrate their color saturation to potential customers, and when he cut lengths of rolled vinyl flooring products ("sheet goods"). No testimony was provided that any of the new asbestos-containing materials handled by Mr. Nacht were friable, and therefore these materials would have generated only low levels of asbestos fiber, if any.

Mr. Nacht also testified that he was exposed to "dust" during inspections he made of nearly all work sites. According to deposition testimony, Mr. Nacht usually inspected between three and five sites per day, and each inspection generally lasted less than 30 minutes. However, the proportion of sites at which Mannington Mills sheet flooring is not known. The purpose of the inspections was to be sure that the job had been completed and cleaned up to Mr. Nacht's satisfaction, as the business owner. Mr. Nacht testified in deposition that he generally insisted that all other trades complete their work before his mechanics began floor installation projects in either new construction or renovations. The asbestos composition of the dust reported by Mr. Nacht is not known, nor the likelihood that this dust contained any asbestos from Mannington Mills products.

According to deposition testimony by Mr. Nacht and an affidavit filed by a former employee of Mr. Nacht, Mr. Daniel Pranzo (employed by Mr. Nacht between 1969 and 1992), Mr. Nacht's business consisted of approximately 60% carpet and 40% tile sales; the majority of the material sold was considered "high end" or "luxury" product. A minority proportion of the business consisted of sales and installation of sheet goods, which was not considered to be a luxury product. Mr. Nacht testified that some of the

sheet goods he sold were produced by Mannington Mills. This allegation was contradicted by Mr. Pranzo in his affidavit, in which he testified that no Mannington Mills products were sold by Mr. Nacht. Between 1963 and 1983, Mannington Mills produced some vinyl flooring stock that incorporated a backing material that contained chrysotile asbestos. If Mr. Nacht did stock and sell Mannington Mills sheet goods, some of those sheet goods may, therefore, have had a backing material that contained asbestos. However, the chrysotile asbestos contained in the backing material would have been bound in a latex rubber matrix, and would not be friable.

Mr. Nacht testified that he cut the sheet flooring goods in order to provide the mechanics only the amount of material needed for a specific installation job. However, Mr. Nacht's former employee, Mr. Pranzo, testified in his affidavit that the flooring mechanics were given the responsibility of performing the "rough cuts", and that Mr. Nacht would only occasionally make these cuts. Making the "rough cuts" to potentially asbestos-containing flooring would have been of short duration, would have occurred intermittently, and would have generated negligible levels of respirable chrysotile fiber, if any were present at all. Such brief, intermittent exposure to chrysotile fibers would not have contributed to any increase in mesothelioma risk.

At the time of his diagnostic workup for mesothelioma, Mr. Nacht was also diagnosed with pleural plaques. Pleural plaques have been associated with asbestos exposure in population studies of autopsy material (i.e., autopsies conducted in populations not defined based on their exposure status) and in occupational and population-based screening programs (Hillerdal 1978; Andrion, Pira et al. 1984; Jarvholm and Sanden 1987; Hillerdal 1994b; Orlowski, Pairon et al. 1994; Bianchi, Brollo et al. 1997; Koskinen, Zitting et al. 1998). Although some researchers question the dose-dependence between asbestos exposure and the extent and progression of pleural plaques (Hillerdal 1978; Hillerdal 1994b; Orlowski, Pairon et al. 1994; Boffetta 1998), the presence of pleural plaques is considered a sensitive indicator of prior asbestos exposure (Orlowski, Pairon et al. 1994; Bianchi, Brollo et al. 1997; Hillerdal and Henderson 1997; Boffetta 1998). While most studies relating pleural plaques to asbestos exposure did not address the role of fiber type, there is some evidence that amphibole asbestos exposure is a stronger risk factor for plaque formation than is exposure to chrysotile (Churg and Vedal 1994; Hillerdal 1994a). Whether or not pleural plaques are precursors for malignancy (carcinoma or mesothelioma) remains an open question (Weiss 1993; Hillerdal 1994b; Bianchi, Brollo et al. 1997; Hillerdal and Henderson 1997; Roggli and Sanders 2000; Koskinen, Pukkala et al. 2002).

CONCLUSIONS

Based on my review, analysis and synthesis of the published epidemiological, occupational health and case-specific information available to me, and assuming that Mr. Nacht actually sold Mannington Mills stock containing chrysotile asbestos, I conclude to a reasonable degree of epidemiological certainty that Mr. Nacht's alleged handling and cutting of rolled vinyl flooring produced by Mannington Mills did not cause Mr. Nacht's mesothelioma. While the literature indicates that substantial numbers of idiopathic pleural mesotheliomas occur, regardless of prior exposure, Mr. Nacht's pleural plaques

suggest that he sustained some past exposure to asbestos, possibly amphibole asbestos. The sporadic potential exposure to low-level chrysotile from handling and cutting Mannington Mills sheet goods containing chrysotile asbestos would have been negligible and of no consequence with respect to his pleural mesothelioma.

Please do not hesitate to contact me if you have questions or require further information.

Sincerely yours,

Kenneth A. Mundt, Ph.D.
Principal and Director of Epidemiology
ENVIRON International Corporation

Case-specific materials relied upon

Description	Date of Document
Nacht Summons & Complaint	10/4/06
Plaintiff's Responses to Fourth Amended Standard Set of Interrogatories and Request for Production of Documents	10/6/06
Deposition of Jack Nacht, Day One	11/21/06
Deposition of Jack Nacht, Day Two	12/4/06
Videotaped Deposition of Jack Nacht	2/6/07
Recordtrak Medical Records	1/24/07
Affidavit of Daniel Pranzo	4/14/07
Canadian Asbestos Information Centre report, supplied in the matter of Cavounis v. Mannington Mills	Undated
Medical Records from Dr. Denise Janus	2/8/07
Expert Report of Brian G. Salisbury, MD	3/11/07
Expert Report of James A. Strauchen, MD	3/12/07

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COLDEN CORPORATION
Occupational Health, Safety & Environmental Consultants

May 31, 2007

Ms. Laura A. Siclari
HOAGLAND, LONGO, MORAN, DUNST, & DOUKAS, LLP
40 Paterson Street
New Brunswick, NJ 08903

RE: Harvey Helfand (In Extremis M07) v. Mannington Mills, Inc.
New York City Asbestos Litigation
Index No. 117176 / 06

Dear Ms. Siclari:

I have reviewed the documents identified below in the matter of Harvey Helfand v. Mannington Mills, Inc. In reviewing these documents, which are identified in Section 1.0, I focused on the following issues:

1. Would handling, including cutting and installing, new Mannington Mills vinyl sheet flooring have resulted in a full work shift, i.e., 8-hour, exposure to airborne asbestos at or above today's Occupational Safety and Health Administration permissible exposure limit of 0.1 fiber per cubic centimeter of air?
2. Did Mannington Mills act responsibly in notifying and/or warning handlers and users of its vinyl sheet flooring products of potential asbestos-related health issues associated with removing such products?
3. To what extent did Mr. Helfand handle floor covering products made by Mannington Mills and to what extent could handling and installing these floor coverings have contributed to his lifetime exposure to asbestos?
4. To what extent did Mr. Helfand experience other occupational and nonoccupational exposures to asbestos and to what extent could these exposures have contributed to his lifetime exposure to asbestos.

Throughout my review of the documents and other materials identified in this report I have relied on my experience as a Certified Industrial Hygienist with over 30 years of experience to frame my thoughts and to shape my analysis of the statements and information I used as the basis for my findings and conclusions. Attached to this report is my curriculum vitae which will provide you a complete history of my professional career.

1.0 Documents and Materials Reviewed

The documents I reviewed relating directly to Mr. Helfand are as follows:

1. Plaintiff's Responses to Defendant's Fourth Amended Standard Set of Interrogatories and Request for Production of Documents, dated November 17, 2006;

2. Deposition upon Oral Examination of Harvey Helfand, pages 1 to 122, January 22, 2006;
3. Deposition upon Oral Examination of Harvey Helfand, Volume II, pages 124 to 258, January 29, 2007;
4. Deposition upon Oral Examination of Harvey Helfand, Volume III, pages 260 to 413, March 9, 2007;
5. Social Security Administration itemized statement of earnings records for Harvey Helfand, dated January 2, 2007;
6. Letter report from E. Neil Schachter, M.D., dated February 12, 2007; and
7. Letter report from Steven H. Dikman, M.D., dated March 14, 2007.

Further, I have reviewed the following documents and materials as they relate to the industrial hygiene aspects of the manufacture and installation of Mannington Mills sheet flooring products:

1. Mannington's internal industrial hygiene monitoring records for worker exposure to airborne asbestos during the manufacture and handling of vinyl sheet flooring; and
2. Third party industrial hygiene monitoring data and reports regarding worker exposure to airborne asbestos during installation of vinyl-asbestos sheet flooring.
3. Mannington's product information bulletins, rolled flooring inserts, and catalog inserts regarding the presence of asbestos in the backing layer of Mannington's vinyl sheet flooring and the safe work practices to be employed in the removal of sheet flooring.

In addition to reviewing these documents and materials I have visited Mannington's vinyl sheet flooring production facility in Salem, New Jersey, and observed the manufacture and handling of vinyl sheet flooring. Although Mannington abandoned the use of a latex-impregnated, chrysotile asbestos backing layer in its sheet flooring products, the manufacturing process using non-asbestos backing remains the same today. Further, I have relied on my personal knowledge of vinyl sheet flooring manufacture and the historical use of asbestos-containing materials in the workplace to arrive at certain conclusions and opinions regarding Mannington Mills and its vinyl-asbestos sheet flooring.

2.0 Worker Exposure to Airborne Asbestos during Installation of Mannington Mills Vinyl Sheet Flooring and Applicable Regulations

The issue of quantifying worker exposure levels to airborne asbestos during the installation of Mannington's vinyl sheet flooring and how those levels compared to applicable guidelines, regulations and standards warrants first a review of the guidelines, regulations and standards that were current at the time of its manufacture and installation.

2.1 Applicable Guidelines and Regulations for Occupational Exposure to Asbestos

Before the passage of the Occupational Safety and Health Act in 1970, there was a consensus guideline for controlling workplace exposure to asbestos that had been established by the American Conference of Governmental Industrial Hygienists (ACGIH) in the 1940s. The

guideline, known as a threshold limit value (TLV), was expressed as millions of particles of airborne dust per cubic foot of air (mppcf). The first TLV for asbestos was 5 mppcf and it was not until the late 1960s that the ACGIH recommended reducing the TLV to 2 mppcf.

During these years, the air sampling method for asbestos yielded dust counts expressed as mppcf. By the late 1960s an improved method for collecting airborne asbestos samples was developed that yielded asbestos results in units of fibers of asbestos per cubic centimeter of air (f/cc). Although there was never a hard conversion factor for converting mppcf to f/cc, a useful conversion factor was 1 mppcf equals 6 f/cc. Thus, 5 mppcf could be considered 30 f/cc and 2 mppcf considered 12 f/cc.

In 1970, with the passage of the Occupational Safety and Health Act, Congress charged the Occupational Safety and Health Administration (OSHA) with setting and enforcing health and safety regulations and standards in the workplace. Prior to 1971 the handling and use of asbestos in general industry and construction was largely unregulated by state or federal agencies. In 1971 OSHA issued its first asbestos standard. This standard set the permissible exposure level (PEL) for airborne asbestos at 12 f/cc as an 8-hour time weighted average (TWA).

In late 1971 OSHA issued a temporary standard that lowered the PEL to 5 f/cc. In mid-1972 OSHA issued a final standard that, first, reinforced this level as the PEL for the next four years and, second, mandated lowering the PEL in mid-1976 to 2 f/cc. The PEL remained at 2 f/cc from July 1976 until 1986, when OSHA lowered it to 0.2 f/cc. The PEL remained at 0.2 f/cc until October 1994, at which time OSHA lowered the PEL to 0.1 f/cc as an 8-hour TWA. This level has remained in effect since then.

When OSHA set the current level it represented the fourth reduction in its PEL since the first PEL was adopted in 1971. Further, when OSHA promulgated this level, it also included in the standard what has been called the "flooring exemption." This exemption permits the removal of flooring materials, which OSHA did not classify as a high risk, in compliance with the procedures developed by the Resilient Floor Covering Institute. These procedures do not require the removal of flooring materials inside negative pressure enclosures. Rather, they require the use of high-efficiency vacuums and wet removal of sheet flooring backing felt, and they prohibit the sanding of flooring, including sheet flooring backing felt, dry sweeping, and mechanical chipping.

It is in the context of this regulatory framework that I reviewed the available airborne asbestos monitoring data and records.

2.2 Independent Air Monitoring Data for Installation of Sheet Flooring

The only reported air monitoring data relating to worker exposure to asbestos during the installation of vinyl sheet flooring were contained in a report prepared in December 1979 by SRI International for the Resilient Floor Covering Institute (RFCI). This report summarized the results of a field study conducted by SRI from December 1978 through June 1979.

The study involved the collection of ten personal air samples on workers engaged in the

installation of vinyl sheet flooring in five private residences. In four of the residences, the installation of the new flooring followed the removal of previously installed sheet flooring. SRI collected and analyzed air samples in accordance with the appropriate National Institute for Occupational Safety and Health method current at the time. This method, which was not specific for asbestos, required counting as asbestos all airborne particles that met the dimensional definition of a "fiber," i.e. a particle 5 micrometers or more in length with an aspect (length to width) ratio of 3 or more to one.

Although the SRI report provided a general description of each residential site and the actual work area, there was no mention of the site conditions at the time of sampling in terms of house-keeping and general cleanliness, nor was there a thorough discussion of the work practices employed by the floor installers. This information, had it been presented and discussed, would have allowed a better understanding and interpretation of the results.

On the basis of their data, the authors concluded that the workers' exposures to airborne asbestos were "very low and well below the OSHA allowable limit." Not only were the measured partial period exposures below the OSHA limit of 2 f/cc current at the time of the study, they were, upon conversion to an 8-hour time weighted averages, below today's PEL of 0.1 f/cc set in 1994.

A subsequent report issued in 1982 by GCA Corporation under contract to the U.S. Environmental Protection Agency discussed these very data, but added no new data. However, the report did characterize the sheet flooring installation process as follows:

> "The only action during installation that disturbs the asbestos felt layer of the flooring is cutting, and this is short in duration and not energy intensive."

Further in the text, the authors stated in their summary of findings:

> "During product installation, the product matrix containing asbestos fibers is disturbed by a limited number of cuts, each of which lasts only 5 to 10 seconds. Total cutting time amounts to 10 to 20 minutes of the total 1 1/2 to 6 hour flooring installation time."

This observation is consistent with my experience and observations of the installation procedures for vinyl sheet flooring.

In the absence of any additional data relating to the installation of sheet flooring, I also considered the largest relevant body of air sampling data available: the Mannington Mills in-house asbestos monitoring program.

2.3 Mannington Mills In-House Asbestos Monitoring Program

Some of the Mannington Mills sheet flooring products manufactured between 1963 and 1983 had asbestos in the bottom, or backing, layer. In early 1976, Mannington initiated an in-house asbestos air monitoring program. Over the next six years Mannington collected dozens of personal exposure and

area air samples for airborne fibers. These samples, which were collected by a Mannington employee in accordance with the sampling method used by OSHA, were analyzed in laboratories accredited by the American Industrial Hygiene Association for the analysis of workplace asbestos air samples.

The data that were particularly relevant to the handling and installation of Mannington's vinyl sheet flooring were those that characterized airborne fiber levels during the unwinding, cutting, and rewinding of rolled felt and sheet flooring through the several steps of the manufacturing process. The data for these activities date back to February 1976 and with few exceptions indicate that the personal exposure levels of workers engaged in these activities and the airborne fiber concentrations in areas where these activities took place were at or below 0.1 f/cc.

I believe that Mannington's data were representative of the actual workplace conditions and worker exposure levels during the manufacture and handling of asbestos-containing vinyl sheet flooring. In fact, the data present a picture of a workplace not only in compliance with OSHA's PEL of first 5 and then 2 f/cc current at the time, but also frequently at or below the lower PELs set in 1986 and 1994.

I believe further that these data also represent potential exposures that are as high as or higher than those of flooring installers if they were to unroll and cut asbestos-backed vinyl sheet flooring for a full 8-hour workday. These data reflect the airborne asbestos levels associated with the manufacturing process when the raw backing layer and flooring were being cut or rolled or unrolled constantly for the duration of the work shift. This is a far more intensive operation than the installation of flooring, when the actual time spent by an installer unrolling and cutting sheet flooring would be only part of the 8-hour workday.

In summary, both the available studies and the extrapolated Mannington workplace exposure monitoring data demonstrate that the installation of vinyl sheet flooring was accompanied by asbestos exposure levels that were below the applicable OSHA PELs throughout the time period that Mannington Mills produced sheet flooring with asbestos in the backing layer. Indeed, the exposures were even below today's PEL 0.1 f/cc.

3.0 Mannington Mills Asbestos Awareness Outreach Program

By the late 1970s it was apparent to Mannington Mills and to the RFCI that the removal of old vinyl-asbestos sheet flooring by dry scraping and/or sanding could lead to worker exposure to airborne asbestos at levels higher than those experienced during installation of new flooring. This awareness led both Mannington and the RFCI to take aggressive steps to not only develop safe work practices for the removal of old flooring, but also to get word of those practices out to floor installers. They did so through the use of widely available written warnings and safe work practices, as well as classroom training programs and training videos.

For example, on May 23, 1977, Mannington alerted its distributors in writing via Bulletin #30 of the potential hazards associated with the removal of existing vinyl-asbestos flooring by sanding. Mannington issued this warning on the basis of information Mannington had developed during the in-

house flooring removal studies it was performing. Subsequent warnings were included in its 1978 product catalog and roll insert sheets.

Mannington's floor removal studies continued beyond the issuance of Bulletin #30 and in February 1978 an internal Mannington memorandum cited a wet method that had been developed for the removal of asbestos-containing backing on existing, in-place sheet flooring. The memorandum specifically mentioned the need to reissue Bulletin #30 on the basis that the new data from the studies indicate that "minimal airborne asbestos fibers" can be expected when wet scraping is employed as the removal method.

In April 1978 Mannington issued Bulletin # 39 to its distributors providing further guidance on the potential hazards of removing existing asbestos-containing vinyl flooring and new guidance on the safe removal of that existing flooring. With reference to its membership in the RFCI and the RFCI's developing role in the safe handling of resilient flooring, the bulletin described the wet method for the removal of the asbestos backing layer of existing sheet flooring before installation of new flooring. From that point forward, Mannington continued to provide the hazard warning and guidance for safe removal in its product catalogs and roll insert sheets.

The issuance of Bulletins #30 and #39 illustrates two significant aspects of Mannington's outreach program. First, Mannington followed good industrial hygiene practice in determining safe and effective methods to remove vinyl sheet flooring by performing air monitoring to evaluate different removal techniques and then using the results to objectively determine the best technique. Second, Mannington acted decisively to notify its customers of the new recommended removal method. These are the actions of a responsible company acting responsibly.

In summary, the documents and materials I reviewed indicate that Mannington Mills took positive and reasonable steps to notify the installers of its vinyl sheet flooring of the potential health hazards associated with the removal of existing asbestos-containing vinyl floor coverings prior to the installation of new Mannington Mills sheet flooring. I believe that Mannington Mills acted responsibly in its efforts to notify the installers of its products and that the written warnings Mannington Mills provided were readily available to the persons who needed to be informed.

During the 1970s, while Mannington Mills was developing and carrying out its asbestos air monitoring and asbestos outreach programs, the federal asbestos regulatory environment was continuing to evolve as well. As described above, by mid-1976 OSHA had lowered its PEL for asbestos from 12 f/cc to 2 f/cc. And it was during this same time period that OSHA carried out its Target Health Hazard Program, a national compliance effort that focused on five occupational health hazards. One of these hazards was asbestos. Between 1972 and March 1976 OSHA industrial hygienists evaluated worker exposure to airborne asbestos in over 650 workplaces. Mannington's flooring plant was not one of those workplaces.

That Mannington Mills was not included in OSHA's program is not surprising. OSHA's focus was on workplaces where asbestos was used as a raw material in the manufacture of asbestos-containing products, e.g., pipe insulation, brake shoes, textiles, etc. Asbestos that already was incorporated or bonded into a product or material used in the manufacture of another product was not viewed in the same way as were the products listed above. The handling and use of backing felts bonded in a latex emulsion were simply not capable of causing worker exposures over the PEL. My personal

experience in OSHA confirmed this when I led an OSHA health compliance inspection of the Armstrong Cork Company flooring plant in Lancaster, Pennsylvania in 1976.

The Environmental Protection Agency (EPA) also focused on asbestos when in 1973 it issued its first National Emission Standard for Hazardous Air Pollutants (NESHAP). Among other requirements, this standard regulated the removal of friable asbestos-containing materials from buildings. (Friable materials can be easily crumbled into loose particles by hand pressure; flooring products are not considered friable.) The EPA acknowledged that the uncontrolled removal of friable materials used in building construction could lead to substantial quantities of asbestos being released into the general environment. In 1979 the EPA published the first of several guidance documents that addressed the control of asbestos-containing materials in schools and, subsequently, all buildings.

The decade of the 1970s was a time of evolving knowledge and awareness regarding the potential hazards posed by the use of asbestos in manufacturing and the removal of asbestos-containing materials from buildings. What had been known about the disease-causing potential of asbestos in medical and research circles was beginning to make its way into the setting of regulatory policy and issuance of occupational health and environmental standards. Whereas the research and industrial hygiene focus in earlier decades had been on the use of raw asbestos in workplaces such as textile mills and insulation manufacturing plants, the focus in the 1970s was on transferring that knowledge to coherent awareness programs and enforceable standards that led to broader and better control of asbestos in all occupational settings.

4.0 Harvey Helfand and Mannington Mills Sheet Flooring

Mr. Helfand's lifelong trade was printing. In the early 1950s he went to a high school in New York City where he learned the printing trade and upon graduation he went to work until 2006 as a printer for various employers in and around New York City.

Although printing was his primary trade, Mr. Helfand pursued a second career as a home remodeler. He did this work not only for the income it provided, but because he loved doing it. In his own words, "[I]t was my pleasure." In some years, home remodeling was his only source of income, but for most of his working lifetime, from 1950 to 2006, home remodeling was something he did on the side while working as a printer. He estimated that over his working career he spent 70 percent of his time in printing and 30 percent in home remodeling. He said his remodeling work was 99 percent residential and focused on basements, kitchens, bathrooms, and other residential spaces; he did not do additions to homes. He also said that he would do the demolition work that necessarily preceded the remodeling work.

According to his testimony, Mr. Helfand "wasn't a big fan of sheet (flooring) goods" and that when he did use sheet flooring, it was at his client's request. Insofar as where he used sheet flooring, he "used it sometimes in a kitchen, or in a laundry room, or something like that or if somebody wanted a simple job." He would buy the sheet flooring rolled and already cut to the approximate size he needed for the space he was remodeling.

Mr. Helfand stated that over the course of his remodeling career he used Mannington Mills sheet flooring on only five occasions. As to when he used Mannington's flooring, he said "I couldn't tell you. I couldn't specifically say I used it in 1960 or whatever. Total over my, you know, varied jobs that I did over the years."

He also testified that when he installed Mannington sheet flooring, he had to make a number of cuts in the flooring as he fitted the sheet to the space being remodeled. He said that while cutting "you get a lot of dust and you get a lot of the garbage, and the fumes and the dust from the Mannington Mills, the linoleum." Earlier in his testimony he said "you got some dust" handling Mannington sheet flooring. Regardless of which of his recollections is more correct, it is my experience that when sheet flooring is cut little or no dust is generated by the process. Rather, the bits and pieces being trimmed from the sheet are what is left behind after cuts are made. Furthermore, there is no indication in Mr. Helfand's testimony that the floor he was handling contained asbestos in the backing layer.

In summary, (i) if Mr. Helfand handled and installed five pieces of sheet flooring made by Mannington Mills between the years 1963 and 1983, (ii) if those five pieces contained asbestos in the backing layer, and (iii) if he did one installation of Mannington product a year in five of the years over that 20-year period, I have calculated that Mr. Helfand's average annual exposure to asbestos in each of those five years would have been less than 0.0001 f/cc. Further, I have calculated that his cumulative exposure over the 20 years from 1963 to 1983 would have been less than 0.000025 f-y/cc. This is an insignificant lifetime exposure.

5.0 Other Exposures to Asbestos-Containing Materials

51. Exposure at Home

Mr. Helfand stated that his first exposure to asbestos came when he was "a kid, eight, nine, ten-years old. We had a house, we had an old house, and we had an old boiler with all the asbestos, you know lined with the white plaster and the pipes wrapped that were constantly cracking and the dust was flying." Although the extent to which Mr. Helfand could have been exposed during his childhood years to airborne dust from the pipe and boiler insulation cannot be determined, his recollection of the insulation and its condition is clear.

The "old house" Mr. Helfand was speaking about was his parent's home at 9802 Foster Avenue in Brooklyn where he lived from 1935 to the early 1950s. Elsewhere in his testimony Mr. Helfand said that he was exposed to and personally handled the asbestos insulation on the boiler in the basement of this house. In his words, "I am sure I was exposed to asbestos because we had a big boiler with the asbestos, you know, enclosed with the white asbestos. We occasionally would make modifications and clean it up."

5.2 Exposure in the Printing Trade

Mr. Helfand described an occupational exposure he had to asbestos during one of his earliest employments in the printing business. While he was still in school, he worked for approximately

a year at Rabin Typographers in lower Manhattan. The lead melting pots used in making the type were lined with asbestos that had been applied as a wet paste. Periodically the hardened asbestos lining would be chipped out and the pots relined. It was the young Mr. Helfand's job to clean up the debris.

5.3 Exposure during Home Remodeling Jobs

When asked if he personally handled any asbestos-containing products during his work in the home remodeling business, the first material he identified was pipe and boiler insulation. He accurately described the insulation as being like a "cast around the boiler and pipes. You know, it's like a cast on your foot." When asked what he did with the insulation during a remodeling job, he said "I just ripped it." Further, he said "(I) Broke it up. You know, I had to break it up to take - like, if we had to remove a boiler or something or remove certain portions of it to remodel, I had to break up that portion."

In regard to Mr. Helfand's exposure to asbestos from pipe and boiler insulation, it is well established in the industrial hygiene and medical literature that the uncontrolled removal of asbestos-containing pipe insulation creates significant concentrations of airborne asbestos. One of the important peer-reviewed articles on the subject appeared in the May-June 1968 issue of the *American Industrial Hygiene Association Journal.* The article, prepared by J. LeRoy Balzer and W. Clark Cooper, M.D., was titled "The Work Environment of Insulating Workers." The field work performed and described in the article was supported by a research grant from the Public Health Service of the U.S. Department of Health, Education, and Welfare. Among the various aspects of the insulating workers' duties the researchers evaluated were the airborne dust levels associated with the different tasks the workers performed. They found that the "tearing out" of existing insulation generated the highest average level of worker exposure among the six tasks they monitored. The average exposure level during tear-outs was 8.9 f/cc.

The authors also described the asbestos-containing materials the insulating workers handled, and most of the materials contained amosite asbestos and to a lesser extent chrysotile asbestos. From a risk assessment standpoint, the scientific literature regarding asbestos disease recognizes a significantly stronger association between exposure to amosite asbestos and mesothelioma than between exposure to chrysotile asbestos and mesothelioma. The American Conference of Governmental Industrial Hygienists, in its documentation for the current Threshold Limit Value for asbestos, states "There is sufficient evidence to show that for a given level of exposure, the risk of developing mesothelioma is far greater with . . . amosite than with chrysotile." During his work as a home remodeler, it is certain that Mr. Helfand was exposed to airborne asbestos when he was ripping out pipe and boiler insulation. It is also possible that while he performed this work Mr. Helfand was exposed at various times to a mixture of asbestos fibers that was predominantly amosite asbestos.

6.0 Conclusion

In summary, it is my opinion that to a reasonable degree of scientific certainty the mixed amosite and chrysotile exposure levels that Mr. Helfand could have experienced while ripping out pipe

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and boiler insulation during his work as a remodeler were several orders of magnitude greater than any exposure level he could have experienced while handling and installing Mannington sheet flooring. Further, the contribution to his lifetime asbestos exposure resulting from the asbestos insulation removal work would be far greater than any contribution that could have come from his handling and installation of five sheets of Mannington Mills flooring.

The conclusions I have drawn and the opinions I have expressed are based on the documents I reviewed as listed above and my professional experience. If ongoing discovery leads to the production of new documents that would bear on the findings and opinions I expressed in this report, I reserve the right to reconsider my findings and opinions and revise the content of this report, as warranted.

Please let me know if you have any questions regarding either my review of the documents and materials identified above or my findings and conclusions.

Respectfully submitted,

COLDEN CORPORATION

Mark F. Durham, CIH
Senior Consultant

Attachment

COLDEN CORPORATION

MARK F. DURHAM, CIH
CURRICULUM VITAE

EXPERIENCE OVERVIEW	Certified Industrial Hygienist with over 30 years experience in occupational and public health hazard management.
PROFESSIONAL CERTIFICATION	Comprehensive Practice of Industrial Hygiene, May 1979 Recertified in 1985, 1992, 1998, and 2003
EDUCATION	MS, Environmental Science/Industrial Hygiene, 1974 Drexel University, Philadelphia, Pennsylvania BA, Biology, 1968 Gettysburg College, Gettysburg, Pennsylvania
SPECIAL EXPERTISE	Asbestos Assessment, Management, and Control OSHA Regulatory Compliance OSHA Litigation Consultation Hazard Awareness Training and Education Programs

EMPLOYMENT HISTORY

Colden Corporation
Senior Consultant
2003 to Present

- Provide consultative and environmental management services to investors, developers, and property owners before, during, and following acquisition of commercial, industrial, residential properties.

Engineering & Fire Investigations
District Manager
2002

- Responsible for opening, staffing, and managing New York City office for national engineering, environmental, and failure analysis corporation.

Clayton Group Services, Inc.
Director, National Accounts
1998 to 2002

- Managed multidisciplinary industrial hygiene evaluations of chemical, biological, and physical hazards in various industrial and commercial workplaces.

- Managed large asbestos abatement projects, including asbestos assessment, abatement design, and removal in 1920's vintage office building under renovation as college dormitory and in 1940's vintage magazine and newspaper printing facility under renovation as telecommunications center.

- Conducted environmental health risk assessment in property adjacent to and damaged by the collapse of the World Trade Center Twin Towers, including identification of asbestos content of airborne dust, determination of decontamination requirements, and oversight of decontamination work.

Galson Corporation
Senior Client Services Manager
1992 to 1998

- Provided expert consultation to New York City Law Department in litigation brought by City against manufacturers of asbestos-containing surfacing materials used in New York City's public buildings.
- Hired, trained, and developed field survey teams for asbestos assessments and lead-based paint testing surveys and lead exposure risk assessments of Department of Defense family housing facilities and priority buildings.
- Managed asbestos and lead-based paint identification and assessment surveys in over 200 million square feet of U. S. Air Force combat, administrative, and housing facilities at over 65 worldwide locations.

Galson Corporation
Industrial Hygiene Division Manager
1990 to 1992

- Responsible for all aspects of industrial hygiene division administration and line management, including profitability and business and client development, in Syracuse, Rochester, New York City, Philadelphia, and San Francisco regional offices.
- Expanded technical service offerings and staff capabilities, including addition of Phase 1 Audits of commercial and industrial properties and indoor air quality evaluations.

Galson Corporation
Regional Office Manager/Corporate Technical Director For Asbestos Services
1985 to 1990

- Named Corporate Vice President for Industrial Hygiene and Asbestos Services (1985).
- Established and managed Philadelphia Regional Office, including AIHA-accredited and NVLAP certified laboratory (1985), and New York City Regional Office (1987).
- Developed written standard operating and quality control procedures for asbestos services, including surveys, design documents, and on-site project monitoring.
- Developed EPA-approved Asbestos Inspector and Management Planner training programs for staff and client personnel.

Galson Corporation
Senior Industrial Hygienist/Manager of Industrial Hygiene Services
1980 to 1985

- Responsible for all of group line management, including profitability, business and client development, technical excellence, and personnel development and retention.
- Developed asbestos survey, abatement design, and project monitoring services as consultant to IBM Corporation, Real Estate and Construction Division.
- Established San Francisco Regional Office to provide industrial hygiene and asbestos management services in northern California.

United States Department of Labor
Occupational Safety and Health Administration
Supervisory Industrial Hygienist and Senior Compliance Officer
1974 to 1980

- Supervised staff of 14 industrial hygienists in Philadelphia Area Office, OSHA's second largest area office in the United States.
- Won significant compliance litigation cases versus steel mills regarding coke oven standard, in foundries regarding lead standard, in refinery regarding OSHA general duty clause for acute chemical exposure fatality, and asbestos products manufacturer regarding willful violations of asbestos standard.
- Performed asbestos compliance surveys in variety of industries, including vinyl asbestos floor tile manufacture, asbestos textile weaving, building products manufacture, and asbestos abatement industries.

United States Air Force
Commissioned Officer
1968 to 1972

- Served as Munitions Maintenance Officer and Nuclear Safety Officer in nuclear weapons fighter/interceptor and missile squadrons at bases in United States and Canada.

Affiliations

- Academy of Industrial Hygiene
- American Industrial Hygiene Association

Related Experience and Activities

- National Asbestos Council: Former Member, Board of Directors; Former Chairperson, Regulatory Affairs and Ethics Committees

CONFERENCES, COURSES, AND PROGRAMS ATTENDED

American Industrial Hygiene Conference and Exhibition
American Industrial Hygiene Association

- *Annual professional organization conference presenting papers and symposia on current issues in industrial hygiene and occupational and environmental health management*

- May 2006, Chicago, IL
- May 2004, Atlanta, GA
- June 2001, New Orleans, LA
- May 1999, Toronto, Canada
- May 1996, Washington, D.C.
- May 1995, Kansas City, MO
- May 1993, New Orleans, LA
- June 1992, Boston, MA
- May 1991, Salt Lake City, UT
- May 1990, Orlando, FL
- May 1989, St. Louis, MO
- May 1988, San Francisco, CA
- June 1987, Montreal, Canada
- May 1986, Dallas, TX
- May 1984, Detroit, MI
- May 1983, Philadelphia, PA
- June 1982, Cincinnati, OH
- May 1981, Portland, OR
- May 1980, Houston, TX
- May 1979, Chicago, IL
- May 1978, Los Angeles, CA

Northeast Regional Industrial Hygiene Conference
Philadelphia/New Jersey/New York Metro AIHA Sections

- *Annual professional conference with focus on special issues in occupational health*

- December 2001, 2002, 2003, 2004, 2005, and 2006, Princeton, NJ

Chemical/Biological Terrorism and Crisis Communication Guidelines for Action
American Industrial Hygiene Association

- *AIHA TeleWeb Virtual Seminar*

- July 200, Philadelphia, PA

Evaluation, Identification, and Remediation of Microbial Contamination

- *MidAtlantic Environmental Hygiene Resource Center*

- October 2002, Philadelphia, PA

Seminar on Legal Issues: Subrogation of Mold Claims

- *White & Williams LLP*
- October 2002, Philadelphia, PA

Mold Assessment and Remediation
Clayton Group Services, Inc

- January 2002, Edison, NJ

Asbestos Project Monitor Course
Asbestos Control Unit, Air Management Services
Department of Health, City of Philadelphia

- June 2000, Philadelphia, PA

Office Ergonomics
Clayton Group Services, Inc.

- March 1999, Seattle, WA

Asbestos Building Inspector Course
Big Apple Occupational Safety Corp.

- April 1998, New York, NY

Tri-Command Environmental Symposium
Education and Training Command, Air Mobility Command, and Space Command, USAF

- *Technical presentations concerning Air Force and Department of Defense environmental programs and policies*

- July 1996, St. Louis, MO

Annual Joint Services Pollution Prevention Conference
Air Force Center for Environmental Excellence

- *Technical presentations concerning Department of Defense, Air Force, Army, and Navy environmental pollution prevention policies and programs*

- August 1996, San Antonio, TX
- August 1995, San Antonio, TX

Environmental Quality Training Symposium
Air Combat Command, USAF

- *Technical presentations concerning Command, Air Force and Department of Defense environmental programs and policies*

- February 1995, St. Louis, MO
- February 1994, St. Louis, MO
- March 1993, Denver, CO

Emerging Environmental Health Issues: An International Concern
National Environmental Health Association

- *Current topics in environmental and public health hazard assessment and control*

- June 1991, Portland, OR

First Syracuse Regional Lead Conference
State University of New York Health Science Center

- *Evaluation and control of community lead hazards*

- February 1997, Syracuse, NY

Annual Technical Conference and Exhibition
National Asbestos Council/Environmental Education Association

- *Current topics in asbestos evaluation and control technologies*

- March 1994, San Diego, CA
- April 1992, Pittsburgh, PA
- September 1989, Indianapolis, IN
- September 1988, Boston, MA
- February 1988, Atlanta, GA
- September 1987, Oakland, CA
- January 1987, Chicago, IL
- February 1986, Baltimore, MD
- February 1985, Dallas, TX
- March 1984, Atlanta, GA

Morgan, Lewis & Bockius, Counselors at Law
Current Developments in Environmental Law

- *Symposium on current and developing regulations, policies, and legal issues*

- November 1987, Philadelphia, PA

Industrial Ventilation
National Institute for Occupational Safety & Health

- April 1978, Cincinnati, OH

Carcinogens in the Workplace
Johnson State University Environmental Symposium

- July 1976, Johnson, VT

Compliance Safety & Health Officer Course
OSHA Training Institute

- April 1974, Des Moines, IL

LECTURES AND PRESENTATIONS GIVEN

American Industrial Hygiene Conference and Exhibition
American Industrial Hygiene Association

- *Presentation given: Asbestos Sampling, Analysis, & Regulatory Update (PDC No. 41)*

- May 1988, San Francisco, CA

- *Presentation given: Rigorous Quality Control Procedures in Analysis of Airborne Contaminants*

- June 1992, Boston, MA

Georgia Tech Research Institute
Atlanta, Georgia

- *Training and education programs for asbestos hazard evaluation and control*

- Course: Inspecting Buildings for Asbestos-Containing Materials/Lecture presented: Pre-Inspection Planning, October 1988
- Course: Advanced Supervision of Asbestos Abatement Projects/Lecture presented: Air Monitoring Update, January 1988
- Course: Managing Asbestos in Buildings/Lecture given: Evaluating and Implementing Response Actions, January 1988
- Course: Asbestos Abatement Project Monitoring/Lecture given: Choosing the Sampling and Data Evaluation Locations for Area and Personal Sampling, March 1986
- Course: Supervision of Asbestos Abatement Contracts /Lecture given: Air Sampling and Analysis at the Completion of an Asbestos Control Project, March 1985
- Course: Supervision of Asbestos Abatement Contracts /Lecture given: Asbestos Control Strategies, March 1985

- Course: Supervision of Asbestos Abatement Contracts/Lecture given: Air Sampling Requirements During Asbestos Control Projects, December 1984
- Course: Supervision of Asbestos Abatement Contracts/Lecture given: Air Sampling Requirements During Asbestos Control Projects, November 1984
- Course: Supervision of Asbestos Abatement Contracts/Lecture given: Air Sampling Requirements During Asbestos Control Projects, November 1984

American Society of Heating, Refrigeration, and Air-Conditioning Engineers (ASHRAE)
Regional and Local Section Meetings

- *Presentation given: Asbestos: Technical, Regulatory, and Liability Issues*

- January 1986, Northeast New York Chapter, Latham, NY
- December 1985, Bi-State (NY & CT) Chapter, White Plains, NY
- October 1985, New York City Chapter Fall Seminar, New York City, NY
- March 1985, North Jersey Chapter, Morrisville, NJ
- February 1985, New York Chapter, New York City, NY
- January 1985, Rochester Chapter, Rochester, NY
- December 1984, Twin Tiers Chapter, Owego, NY
- August 1984, Region I Technical Conference, Corning, NY

American Society of Plumbing Engineers (ASPE)
National and Local Section Meetings

- *Presentation given: Asbestos-Containing Materials in Buildings: Health Concerns*

- February 1987, Delaware Valley Chapter, Philadelphia, PA
- November 1986, 1986 ASPE National Convention, Miami, FL
- November 1985, Washington, DC, Chapter, Fairfax, VA
- November 1984, 1984 National Convention, Chicago, IL

Drexel University, Philadelphia, PA
Asbestos Hazard Evaluation & Abatement Workshop

- *Presentations given: Air Monitoring & Sampling Procedures and Evaluation of Air Sampling Data During Asbestos Abatement Projects*

- September 1986, Philadelphia, PA

City of Philadelphia Public Health Department
Asbestos Project Certification Course

- *Presentation given: Response Actions When Air Sampling Results Exceed Limits*

- March 1990, Philadelphia, PA

Prentice Hall Law and Business Seminars
Coping with Asbestos in Commercial and Public Buildings

- *Presentations given: (1) How To Determine If You Have A Problem & Choosing Asbestos Response Actions, (2) How To Plan and Implement an Asbestos Operations & Maintenance Program, (3) Asbestos Regulations: Clean-Up Obligations & Proposed Regulations*

- April 1990, New York City, NY
- October 1989, New York City, NY
- June 1989, New York City, NY
- October 1988, New York City, NY
- January 1988, New York City, NY

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